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The Case | Atrial fibrillation after a soccer match

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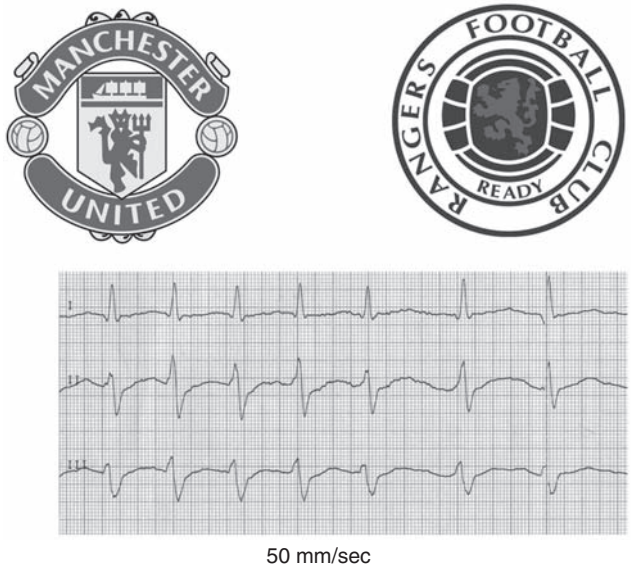

Figure 1 | These symbols were directly related to weakness in this patient. Limb leads of the electrocardiogram (I, II, III) show atrial fibrillation; the QT interval is prolonged (right).

A 41-year-old Scot visited Berlin on a business trip. On the evening before his appointments, he watched a soccer match between Manchester United and the Glasgow Rangers (Figure 1). The game ended in a frustrating 0:0 outcome for the Glasgow Ranger fan. He consumed three beers and a pizza during the match. Back in his hotel, he observed palpitations, a rapid heart rate, and profound weakness. His quadriparesis made calling an ambulance extremely difficult. His past medical history was uneventful and he denied excessive alcohol or drug intake. However, he reported a somewhat similar episode 3 weeks earlier, for which he had not sought medical attention. His blood pressure was 154/65 mm Hg, respiratory rate 20/min, and the heart rate was irregularly irregular, at about 100 beats/min. No pulse deficit was reported, although the first heart sound varied in intensity beat to beat. No extra sound or elevated filling pressures were observed. The electrocardiogram (50 mm/s) showed atrial fibrillation with a prolonged QT interval (Figure 1). Arterial blood gas analysis revealed pH 7.43, $PaCO_2$ 35 mm Hg, and PaO_2 54 mm Hg. The HCO_3 level was 24 mmol/l. Serum Na was 140 mmol/l, Cl 111 mmol/l, glucose 141 mg/dl, lactate 17 mg/dl (normal), and K 1.6 mmol/l. The troponin level was normal, creatine kinase MB was modestly elevated, and the ethanol level was in the 'sober' range. A portable chest roentgenogram was unremarkable.

What is the likely cause of this patient's hypokalemia?

SEE NEXT PAGE FOR ANSWERS

The Diagnosis | Hypokalemic thyrotoxic periodic paralysis (TPP)

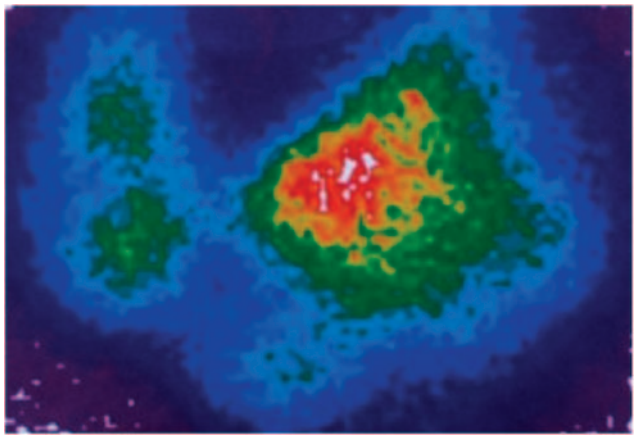

Figure 2 | Tc99 scintigraphy shows an active nodule in the left upper lobe of the thyroid gland.

The patient's thyroid stimulating hormone was <0.01 μIU/l, free T3 17.4 pg/dl (normal >5), and free T4 4 pg/dl (normal <5.9). The emergency-room physicians did not take care to measure bladder urine K level. The urine K level in a spot specimen, after infusion of 100 mmol potassium chloride, was 59 mmol/l. At that time the serum K level had increased to 4.8 mmol/l and the weakness resolved. Scintigraphy revealed a 'hot' thyroid nodule (Figure 2). Methimazole and propranolol resulted in reversion to sinus rhythm. Non-selective beta-blocker treatment decreases K requirements and minimizes subsequent serum potassium disturbances.[1]

Serum K disturbances *almost always* feature acid–base disturbances. The potassium-related periodic paralyses are an exception.[2] Nonetheless, Lin *et al.*[2] underscored the importance of diagnosing metabolic acid–base disturbances in hypokalemic patients. Our patient had normal pH, $PaCO_2$, and HCO_3 values, which made total-body K depletion unlikely. The thyroid stimulating hormone value tipped the diagnosis. The admitting physician did not judge the thyroid gland as abnormal; however, the laboratory helped in this regard. Thyrotoxic periodic paralysis (TPP) is found in patients of Chinese, Vietnamese, Korean, Filipino, Native American, and Hispanic ancestry, but is apparently less common in Europeans.[3] Nonetheless, a recent report emphasizes that TPP is not that uncommon in Western countries.[4] Early diagnosis and prompt treatment prevent life-threatening complications associated with hypokalemia and muscle weakness. Assaying thyroid function in patients with hypokalemic paralysis helps distinguish TPP from other forms of hypokalemic periodic paralysis. Hypokalemia and muscle paralysis result from a sudden intracellular potassium shift. As a result, the clinical features of TPP are subtle. Potassium supplementation is helpful for the recovery of muscle weakness, but careful monitoring is required. Beta-adrenergic blockers can ameliorate and prevent recurrence of the paralytic attacks. Definitive control of hyperthyroidism is mandatory. The hypokalemic mechanism involves increased sodium–potassium ATPase activity and enhanced insulin response in patients with TPP. Ryan *et al.*[5] very recently identified a previously unreported gene encoding an inwardly rectifying potassium (Kir) channel, Kir2.6. This channel resembles Kir2.2, is expressed in skeletal muscle, and is transcriptionally regulated by thyroid hormone. Kir2.6 mutations were found in 33% of their unrelated TPP patients. Those patients were predominantly from Singapore, while in patients from Hongkong and Thailand Kir2.6 mutations were hardly observed. This state of affairs suggests that we will learn of additional channels regulated by the thyroid hormone. We conclude that TTP can occur anywhere and that vigilance can help in prevention of serious complications.

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